International Journal of Advances in Medicine
Dwipayana PA et al. Int J Adv Med. 2022 Jul;9(7):835-839
http://www.ijmedicine.com
pISSN 2349-3925 | eISSN 2349-3933
DOI: https://dx.doi.org/10.18203/2349-3933.ijam20221712

Case Report

Asymptomatic isolated intramuscular cysticercosis in diabetic patient: a case report

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Received: 12 May 2022
Accepted: 02 June 2022

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ABSTRACT

Cysticercosis is an endemic disease in multiple areas of the world, caused by ingestion of embryonated eggs of *Taenia solium*. We reported a case of a 66-year old diabetic male presenting with decrease of consciousness and "rice grain" calcification in both hemithorax and neck soft tissue region, without findings of cysticercosis in computed tomography (CT) scan. Cysticercosis most commonly affects the central nervous system, causing neurocysticercosis (NCC), while solitary intramuscular cysticercosis without involvement of the central nervous system is rare. Treatment of cysticercosis is indicated for symptomatic patients, including NCC surgery is indicated in patients with neurovascular compromise caused by growth of cyst. Recognizing the signs and symptoms of cysticercosis is crucial for diagnosis and treatment in endemic areas where *Taenia solium* is prevalent.

Keywords: Intramuscular cysticercosis, Diabetes mellitus, Taenia solium

INTRODUCTION

Cysticercosis is an endemic disease in developing countries. Human cysticercosis is found worldwide, especially in areas where porcine cysticercosis is common. Both taeniasis and cysticercosis are most often found in rural areas of developing countries with poor sanitation, where pigs roam freely and eat human feces.[3]

Human cysticercosis can result in devastating effects on human health. The larvae (cysticerci) may develop in the muscles, skin, eyes and the central nervous system. The clinical manifestations of patients vary upon the sites of larval encystments.[2] There have been reports of a few cases of intramuscular cysticercosis that did not affect any other organ.[1] Cysticercosis infect humans when they swallow eggs that are excreted in the stool of people with the adult tapeworm. This may happen when humans drink water or eat food contaminated with tapeworm eggs or put contaminated fingers in their mouth.

Cysticercosis is not spread by eating undercooked meat. However, people get infected with tapeworms (taeniasis) by eating undercooked infected pork. People who have tapeworm infections can infect themselves with the eggs and develop cysticercosis (this is called autoinfection). They can also infect other people if they have poor hygiene and contaminate food or water that other people swallow. People who live with someone who has a tapeworm infection in their intestines have a much higher risk of getting cysticercosis than other people.[3,4]

CASE REPORT

A 66-year-old male patient presented to the emergency department with agitation and confusion this morning. Symptoms appeared suddenly after patient was sleeping before. Patient also complained of fever today. His past medical history was routinely controlled hypertension treated with valsartan 1×80 mg and no history of other chronic diseases (diabetes mellitus, chronic kidney disease, stroke, liver disease).

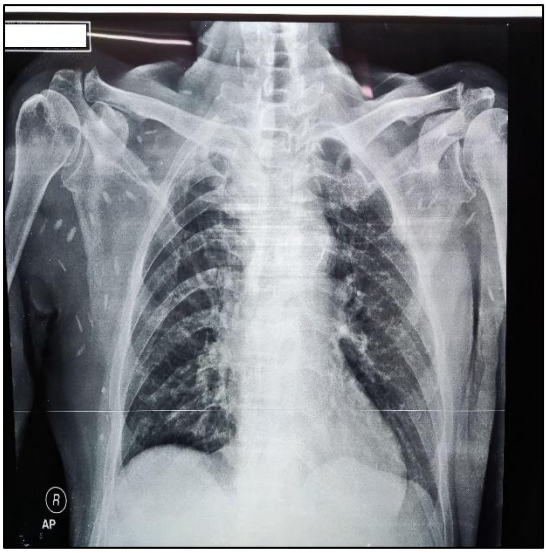

Figure 1: Anteroposterior (AP) chest X-ray with rice grain calcification appearance.

On physical examination, his blood pressure was 154/95 mmHg, heart rate 117 bpm, respiratory rate 20 per minute, and temperature was 38.5°C. A Glasgow coma scale of 10 (E2, M4, V4) was documented on presentation.

No signs of other neurological deficits were present. On examination and palpation of the muscle and subcutaneous tissues, no nodules were palpable. Routine laboratory findings were: haemoglobin 12.5 g/dl, white blood cell 12.680/µl, haematocrit 36.7%, thrombocyte count 252.000/µl. Differential count shows increase in neutrophil and monocyte, and decrease in lymphocyte. AST 23 U/l, ALT 20 U/l, BUN 43 mg/dl, serum creatinine 1.8 mg/dL, natrium 127 mmol/l, potassium 3.7 mmol/l. Urinalysis showed leukocyte esterase (-), protein +1, glucose +4, blood +1, and bacteria (+). Random blood glucose sampling showed 'high' mg/dl. After starting intravenous insulin, blood glucose was decreased to 592 g/dl. His blood gas analysis was on normal range.

An anteroposterior (AP) chest X-ray was performed showing normal lung, aortosclerosis, and suspected cysticercosis ('rice grain calcification') in bilateral hemithorax and neck soft tissue region. Non-contrast CT scan of the brain showed subacute ischemic cerebral infarction in right frontal lobe, left corona radiata, and left basal ganglia, and also chronic ischemic cerebral infarction in right basal ganglia, right corona radiata, frontal lobe, and left head of caudate nucleus, and mild brain atrophy. No radiologic features of neurocysticercosis (NCC) were found in the brain from the CT scan.

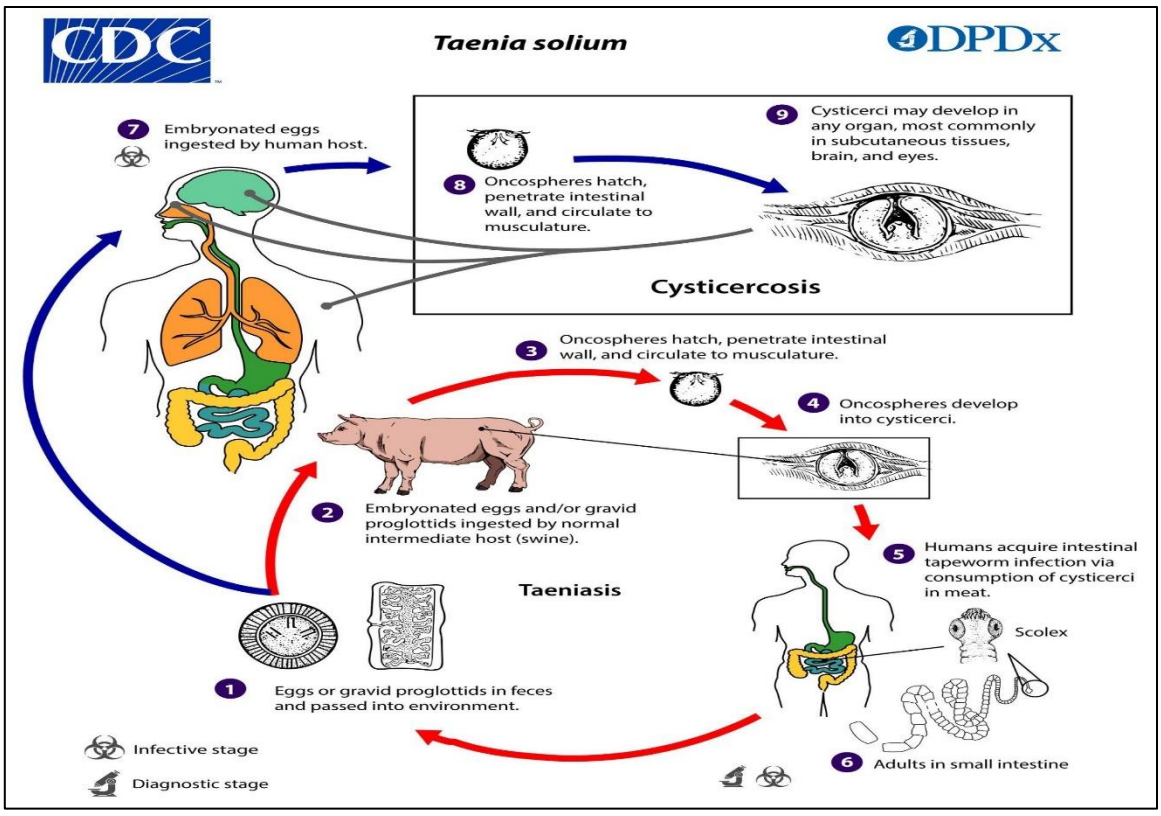

Figure 2: *T. solium* life cycle.[3]

We established the diagnosis of type 2 diabetes mellitus with hyperglycemic hyperosmolar state (HHS), ischemic stroke, asymptomatic cysticercosis, and stage I hypertension. Patient was treated with intravenous insulin according to HHS algorithm. Intravenous ceftriaxone 2 g BID, methylprednisolone 62.5 mg BID, and citicoline 500 mg BID. was given. Patient was also treated with oral albendazole 400 mg QD. After 24 hours of insulin

treatment, his blood glucose dropped to normal range, and intravenous insulin was changed to subcutaneous basal bolus insulin. Patients' consciousness returned to normal following the reduction of his blood sugar. Over the course of treatment, patient's HbA1c was measured (>15.0%). No parasites or worm eggs were found on the fecal analysis. Patient was discharged after 8 days of treatment.

DISCUSSION

Cysticercosis is an infection caused by the larval form of the intestinal tapeworm, *Taenia solium*. Infection occurs after ingestion of ova containing viable oncospheres that invade the intestines, enter the vasculature and develop in the tissues.[5] This infection can have quite serious effects, especially if it involves the central nervous system (NCC) and visual organs (ophthalmic cysticercosis) with the former more commonly found than ophthalmic cases.[7] Note that cysticercosis is acquired from the fecal-oral route (ingestion of eggs), not via the ingestion of cysticerci in undercooked pork, which is associated with intestinal taeniasis.[3]

Cysticercosis develops when, following ingestion of *T. solium* eggs, *T. solium* larvae migrate and become encysted.[8] Cysts are ingested and The outer shell of the cyst is broken by the gastric secretions, leaving behind the head of the parasite known as scolex. The scolex has four cup-shaped suckers and a double row of prominent hooks (rostellum) that help in anchorage to the small intestinal mucosa. After attachment, the scolex proliferates and becomes a mature tapeworm by 5-12 weeks. The adult tapeworm then sheds eggs and proglottids into human feces that can contaminate the pig food supply. Eggs are thickly shelled and hence are not destroyed in the soil for a long time. When pigs or humans ingest the eggs, the gastric secretions break the outer wall of eggs. The oncospheres are released which attach and penetrate the intestinal wall. On the surface of oncospheres, hooks and suckers are present which helps in attachment and penetration of the intestinal wall. Oncospheres enter into the mesenteric venules and via bloodstream lodge in various tissues, and develop into cysts.[13] Cysts may lodge anywhere in the body. Brain, eyes, subcutaneous tissue and muscles are more commonly affected. Humans and pigs are intermediate hosts of larvae of *T. solium*. Human feces that contain eggs *T. solium* can accidentally contaminate plants so that humans ingest them. This contamination can occur especially in areas with poor sanitation.[6]

Taenia solium was endemic in multiple areas of the world, including sub-Saharan Africa, Latin America, Southeast Asia, India, and China.[9] In Indonesia, the highest prevalence for cysticercosis were found in North Sumatra, Bali, and Papua. The seroprevalence of cysticercosis in Bali ranged from 5.2 to 21% during 1981-1997. Since then, cysticercosis cases have decreased dramatically in Bali (only 2 sero-positive cases were reported among 660 people during 2002-2009), which may be caused by improvement of sanitation and sustainable public health education. The risk factor of cysticercosis in bali includes consumption of 'pork lawar', an uncooked dish with minced pork and fresh pig blood.[10]

Cysticercosis most commonly affects the central nervous system (97.46%), followed by ophthalmic (1.4%), skin and soft tissues (1.14%).[11] Subcutaneous tissue involvement may present as palpable nodules, and diffuse muscular involvement may present as myalgia, weakness, or pseudohyperthropy.[12] Solitary intramuscular cysticercosis without involvement of the central nervous system is rarely found and usually not fatal, even though sometimes it can cause compression of neural structures causing compressive neuropathy and fibrosis of muscle fibers causing Volkmann's contracture.[13]

Multiple serological tests are available for the diagnosis of cysticercosis, such as enzyme linked immunosorbent assay (ELISA), complement fixation test, and enzyme linked immuno transfer blot (EITB). EITB has a specificity of 100%, sensitivity of 98% for multiple cerebral lesions, and sensitivity of 60-85% for patients with single cystic lesions. Hematological examinations are non-specific, where ESR and eosinophil count may be increased, indicating helminthic infection. Transient eosinophilia can be seen in the early course of the disease which is associated with migration of the larvae through the blood or tissue. Definitive diagnosis of cysticercosis requires histopathological studies to visualize the parasite. Alternatively, fine needle aspiration cytology can be used as alternative to histopathologic examination.[13]

Radiologic examination of cysticercosis depends on the stage of the infection. Calcified cysts are apparent, while cysticerci rarely shows on plain radiographs. From CT scan, multiple cysts are seen as multiple seed-shaped elliptical calcifications, called 'starry-sky' or 'rice grain' appearance. Ultrasonography may show intramuscular cyst with eccentric echogenic scolex. The gold standard for diagnosis of intramuscular cysticercosis is MRI which is superior than CT scan in evaluating and detecting stage of disease and perilesional edema.[13]

Antihelminthic treatment for cysticercosis is indicated for symptomatic patients with multiple live cysticerci, and will not benefit patients with calcified cysts. Destruction of live cysticerci causes inflammatory response, which corticosteroid is indicated in combination with anthelmintic treatment. Conventional anticonvulsant therapy is given in NCC associated seizure disorders.[13] The recommended anthelmintic for NCC is albendazole 15 mg/kg/day for 10 days in patients with 1-2 viable cysts and albendazole plus praziquantel 15 mg/kg/day for 10 days in >2 viable cysts.[14] This patient was given oral albendazole 400 mg QD for 10 days. For isolated intramuscular cysticercosis, the reported treatment options ranged from medications, surgery, or watchful waiting. No specific treatment is usually indicated for isolated skeletal muscle or subcutaneous cysticercosis. Surgical removal may be

required when there is neurovascular compromise caused by growth of the cyst. Some studies also reported praziquantel 50 mg/kg/day for approximately 3 weeks for treatment of cysticercosis.[12,15]

Parasitic diseases have been consistently reported in association with diabetic patients. The course of the infections is also more complicated in this patient group. Moudgil et al reported that 10.8% (8/74) of the patients with cysticercosis were hyperglycemic. The overall prevalence of diabetes with toxoplasmosis, hydatidosis, and cysticercosis patients' samples was highest with hydatidosis 11.49% (10/87) followed by cysticercosis and toxoplasmosis. Most studies show decreased functions (chemotaxis, phagocytosis, and killing) of diabetic polymorphonuclear cells and monocytes/macrophages compared to cells of controls.[16] There is a large spatial overlap between intestinal parasites and diabetes distribution, and the pathogenic mechanisms of both diseases suggest that they might influence each other; however, few studies on the occurrence of intestinal parasites in diabetic individuals were made until now.

In this case we confirmed cysticercosis inadvertently by doing a chest X-ray examination during an emergency ward visit due to decrease of consciousness. Initially, we suspected that NCC had occurred but CT scan of the head showed no sign of NCC. MRI of the head with contrast is the best-known modality in detecting the initial phase of NCC, other investigations that can be used to support the diagnosis are immunodiagnostic tests. The examinations use an enzyme-linked immunosorbent assay (ELISA) and Enzyme linked immunoelectrotransfer blot technique (EITB), but due to limited facilities and instruments, radiologic and serological examinations such as MRI, ELISA, or EITB cannot be performed.[6]

CONCLUSION

Cysticercosis is a parasitic infection caused by ingestion of embryonated eggs of *Taenia solium* by a human host. *Taenia solium* was endemic in multiple areas of the world, including Indonesia, where the highest prevalence for cysticercosis were found in North Sumatra, Bali, and Papua. Cysticercosis mainly affects the central nervous system, causing NCC. Soft tissue involvement can be asymptomatic or symptomatic if compression of adjacent neural structure occurs or fibrosis of the muscle. The gold standard of diagnosing cysticercosis is histopathological visualization of the parasite. EITB is also recommended as serologic testing for cysticercosis. Asymptomatic intramuscular cysticercosis requires no specific treatment. Parasitic infection in diabetic patients is well recognized, while the number of reported cysticercosis cases in diabetic patients is scarce. Despite the decreasing number of cysticercosis in Bali, the widespread consumption of pork meat still entails risk of cysticercosis. Recognizing the signs and symptoms of cysticercosis is crucial for diagnosis and treatment in endemic areas where *Taenia solium* is prevalent.

ACKNOWLEDGEMENTS

We would like to thank Ketut Suryana as chairman of Internal Medicine Department, Wangaya General Hospital, Denpasar, Bali, Indonesia for reviewing our work and general support.

Funding: No funding sources
Conflict of interest: None declared
Ethical approval: Not required

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Cite this article as: Dwipayana PA, Adrian SJ, Diyana MW. Asymptomatic isolated intramuscular cysticercosis in diabetic patient: a case report. Int J Adv Med 2022;9:835-9.